further depress the patient whose symptoms are due to barbiturates or other depressants.

Naloxone will promptly (within 1 to 2 minutes) and dramatically reverse the respiratory depression but its antidotal action lasts only 2 to 3 hours. The depressant effects of the narcotic-like drugs last from 24 to 48 hours. Therefore it is important to monitor the patient continuously during this period and administer repeated doses of naloxone before serious respiratory depression can reoccur.

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REFERENCES

Ginsburg C: Lomotil intoxication. Am J Dis Child 125:241, 1973 Aronow R, Paul SD, Woolley PV: Childhood poisoning: An unfortunate consequence of methadone availability. JAMA 219: 321-324, Jan 17, 1972

Factors Affecting Lung Maturation

THE IDIOPATHIC RESPIRATORY DISTRESS SYNDROME (IRDS) is a disease of premature infants. It accounts for approximately 25,000 deaths per year in this country. Perhaps the disease should no longer be termed "idiopathic." According to the work of such investigators as Avery, Mead, and Gluck, it appears to proceed from a deficiency of surfactant resulting either from a delay in maturation of the alveolar lining cells producing surfactant or from a defect in release of the substance from the cell.

Using the lecithin sphyngo-myelin (L/s) ratio determination or the bubble stability test on amniotic fluid, it now has become possible to routinely determine the extent of maturation of the human fetal lung. Several conditions including maternal heroin addiction, intrauterine growth retardation, and perhaps amniotic membranes ruptured longer than 16 hours probably cause an accelerated production or release of surfactant.

Liggins showed that maturation of fetal lamb lungs could be accelerated by the antepartum administration of glucocorticoids. Liggins and Howie have published evidence that betamethasone injected into women in premature labor at least 48 hours before delivery decreases the incidence of IRDs in infants born before 32 weeks' gestation. Baden and other investigators in Montreal found no decrease in the incidence of hyaline membrane disease in infants receiving corticosteroid therapy after delivery.

However, more studies are necessary before the routine use of corticosteroids in premature labor can be recommended. In extreme situations, when delivery is inevitable before 32 weeks gestation

and the L/s ratio is low or the bubble stability test negative, administration of betamethasone might be considered.

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REFERENCES

Liggins GC, Howie RN: A controlled trial of antepartum glucocorticoid treatment for prevention of the respiratory distress in premature infants. Pediatr 50:515-525, Oct 1972

Baden M, Bauer CR, Colle E, et al: A controlled trial of hydrocortisone therapy in infants with respiratory distress syndrome. Pediatr 50:526-534, Oct 1972

Hypothalamic Factors

HYPOTHALAMIC NEUROHUMORAL control of pituitary hormone secretion has been established. There are at least nine neurohumors, with three now synthesized. Adrenocorticotropic hormone (ACTH), thyroid stimulating hormone (TSH), luteinizing hormone (LH) and follicle stimulating hormone (FSH), are responsive to hormones from target glands which inhibit further secretion by action exerted on the pituitary, the hypothalamus, or both. Hypothalamic inhibitors for growth hormone (GH), melanocytic stimulating hormone (MSH) and prolactin compensate for the absence of negative feedback products from their target organs.

Thyroid releasing hormone (TRH), a tripeptide that has been synthesized, is effective when administered either orally or parenterally. It stimulates TSH and prolactin release and is inhibited by thyroid hormones. Clinically, TRH can be used to differentiate primary hypothalamic and pituitary causes of hypothyroidism, confirm Graves' disease, measure TSH reserve and increase milk production in cows.

Luteinizing releasing factor (LRF), a decapeptide that also has been synthesized, is capable of stimulating-gonadotropic secretions. A specific follicle releasing factor (FRF) has not been identified. The prepubertal hypothalamus is inhibited by low levels of circulating sex steroids. At puberty, steroid-mediated hypothalamic maturation leads to increased LRF production and increased pituitary stores of gonadotropin. LRF stimulation then leads to a heightened release of LH and FSH. Elevated sex steroids can alter pituitary responsiveness to LRF. Clinically, some patients with hypogonadotropic hypogonadism have been found to have a deficiency of LRF rather than of LH or FSH. Prolonged therapy with LRF may be helpful in treating oligospermia and azospermia. An LRF antagonist might be useful in birth control.

Somatotropin release inhibiting factor (SRIF),